REMARKS

These remarks are in response to the Office Action mailed December 28, 2006. Claims 20 and 28 have been canceled without prejudice to Applicant's right to prosecute the canceled subject matter in any divisional, continuation, continuation-in-part, or other application. Claims 15-17, 25 and 31 have been amended to remove recitation of non-elected species, to incorporate the subject matter of claim 20 and 28 and to correct antecedent basis following the claim amendments. No new matter is believed to have been introduced.

I. ELECTION/RESTRICTION

Applicant confirms election of species directed to NT69L.

II. DRAWINGS

Replacement drawings are submitted concurrently herewith. Please substitute the attached drawings for those currently in the application.

III. CLAIM OBJECTION

Claims 15-17, 19, 23, 25, 30 and 31 stand objected to because the presence of non-elected species. Applicant has amended the currently claims to recite the elected invention for the convenience of the Examiner. Applicant reserves the right to rejoin the non-elected species upon indication of allowable subject matter.

IV. REJECTION UNDER 35 U.S.C. §112

Claims 15-18, 20-26 and 28-30 stand rejected under 35 U.S.C. §112, second paragraph, as allegedly being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

In particular, the term "modulating" is allegedly indefinite in claims 15-18 and 20-23. The claims have been amended to indicate that the modulating is increasing sensorimotor gating. Applicant believes that the foregoing amendments overcome the rejection.

Claims 24-26 and 28-30 for allegedly lacking the step indicating the inhibition of serotonin-2A and/or alpha-1 receptor mediated neural function. This rejection is moot with respect to canceled claim 28. Claim 24, upon which claims 25-26 and 29-30 depend, has been amended to overcome the rejection.

Claims 16-18 and 20 are allegedly indefinite for recitation of "improving", "symptoms" and "subject". Applicant has amended the claims to set forth that the improvement is as compare the subject without the treatment. The "symptoms" have been amended to indicate that they are associated with reduced prepulse inhibition. By reference to "subject" in the specification one of skill in the art would be apprised of the scope of the term. Thus, Applicant submits that the term is not indefinite.

V. REJECTION UNDER 35 U.S.C. §112, FIRST PARAGRAPH

Claims 15-16, 21-23 and 29-34 stand rejected under 35 U.S. C. §112, first paragraph, because while the specification is enabling for a method for administration of a neurotensin agonist to a subject with reduced pre-pulse inhibition, thereby promoting an elevation in the PPI response inhibition, the specification

allegedly does not enable a method of modulating sensorimotor gating in a subject having a neuropsychiatric disorder, such as bipolar disorder, by administering any neurotensin agonist, thereby increasing PPI. Applicant respectfully traverses this rejection with respect to the amended claims.

Prepulse inhibition is a type of sensorimotor gating recognized in the art. Sensorimotor gating disorders in subjects with neuropsychiatric disorders is measured by prepulse inhibition measurements. Thus, Applicant submits that the scope of the claims is fully supported by the specification and one of skill in the art.

Furthermore, the claims have been limited to the elected species and thus do not encompass "any" neurotensin agonist (Applicant reserves the right to rejoin non-elected species).

The Office Action states that Ni et al. teach that the serotonin-2A gene is unlikely to play a role in the "genetic susceptibility to bipolar disorder." Applicant respectfully submit that the invention is not regulating or treating the genetic expression of serotonin-2A. Thus, the teaching of Ni et al. is misplaced with respect to the claimed invention.

The Office Action further alleges that the lack of a valid animal model is a major limitation for considering the application of the claimed invention to bipolar disorders. Applicant respectfully traverses this rejection.

The Examiner is respectfully reminded that the claims need bear only a reasonable correlation to the disclosure and example. The issue of "correlation" is related to the issue of the presence or absence of working examples and the relationship between *in vitro* or *in vivo* animal model assays and a disclosed or a claimed method of use. *Training Materials for Examining Patent Applications with*

Respect to 35 U.S.C. Section 112, first paragraph -- Enablement

Chemical/Biotechnical Applications. An *in vitro* or *in vivo* animal model example in the specification, in effect, constitutes a "working example" because that example "correlates" with a disclosed or claimed method invention. Applicant has provided data from an animal model characterized as having a reduced prepulse inhibition. Reduced prepulse inhibition is also recognized as being present in neuropsychiatric disorders. Accordingly, methods of treating diseases or disorders associated with a deficit in prepulse inhibition (such as, for example, Schizophrenia) bear a reasonable correlation to the claimed subject matter and support in the specification.

For at least the foregoing reasons, Applicant respectfully requests withdrawal of the rejections set forth above.

VI. REJECTION UNDER 35 U.S.C. §102(b)

Claims 15-18, 24-26 and 31 stand rejected under 35 U.S.C. §102(b) as allegedly anticipated by Feifel et al. (J. Pharmacol. & Exp. Therap. 288:710-713, 1999). Applicant respectfully traverses with respect to the claims as amended.

Feifel et al. do not teach or suggest NT69L, as currently elected in response to the restriction requirement. Furthermore, with reference to claim 17, Feifel et al. do not teach or suggest a combination therapy with other psychotropic drugs. As set forth in the specification typical and atypical psychotropic drugs act by binding to the dopaminergic receptor (typical) or to the serotonin-2A and dopaminergic receptor (atypical). In contrast, NT69L binds to neither of these receptor, yet is capable of inhibiting serotonin-2A and/or alpha-1 receptor mediated neural function. Similarly,

Feifel et al. do not teach or suggest a serotonin-2A-, dopaminergic-receptor independent activity of NT69L.

For at least the foregoing reasons, Applicant submits that Feifel et al. do not teach or suggest the claimed invention. Accordingly, Applicant respectfully request withdrawal of the rejection.

Respectfully submitted,

BUCHANAN INGERSOLL & ROONEY LLP

Date: March 28, 2007 By: _____

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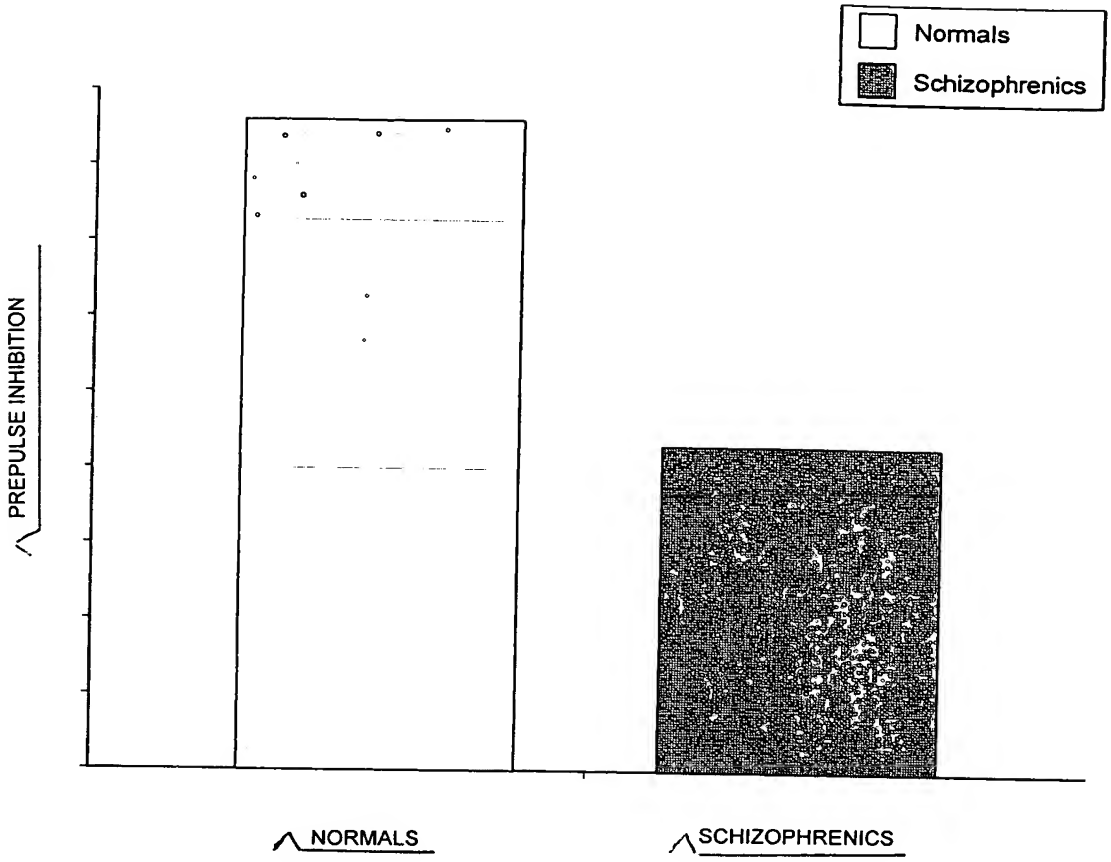

Figure 2

PPI is reduced in Schizophrenia patients

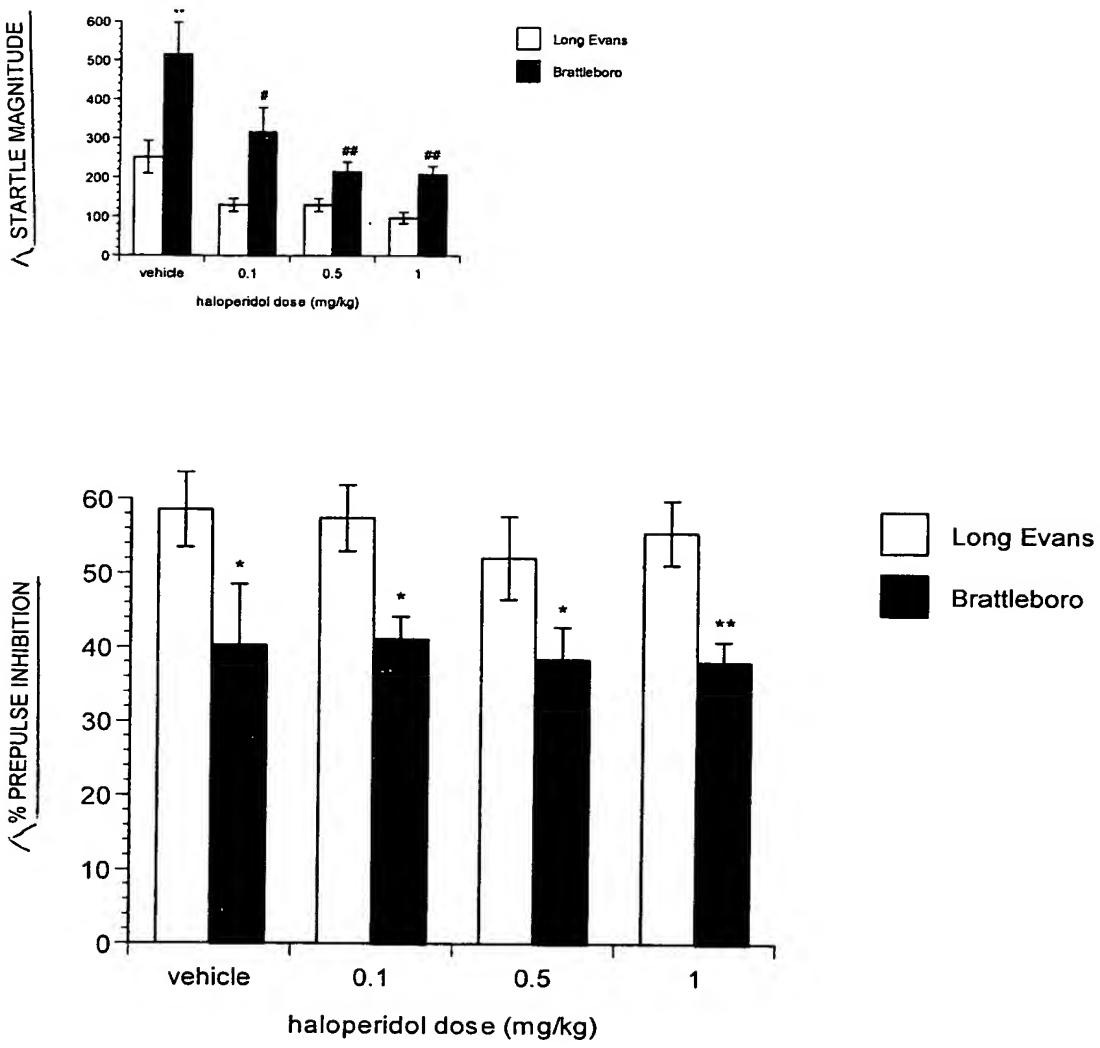

Figure 7

Haloperidol does no effect PPI deficits in BB rats

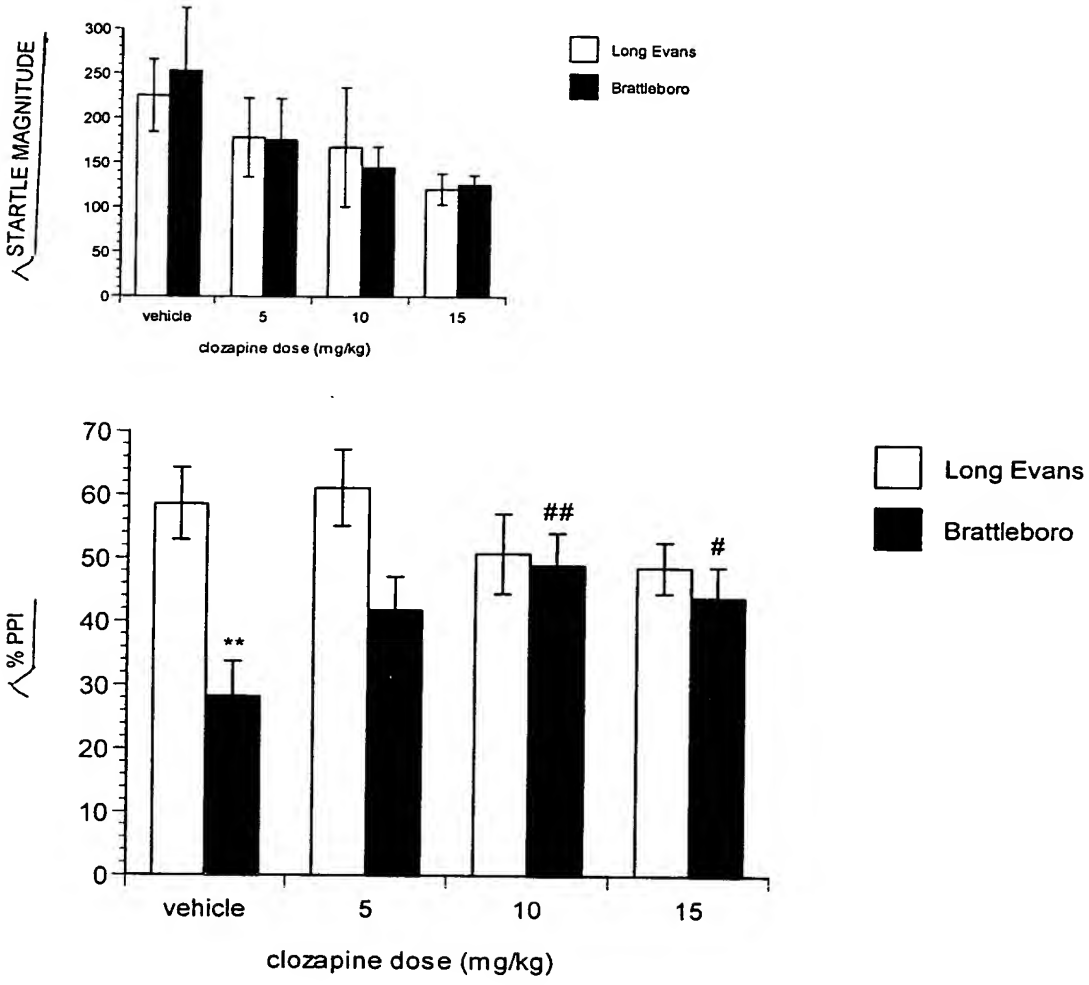

Figure 8

Clozapine, dose-dependently increased PPI in BB rats

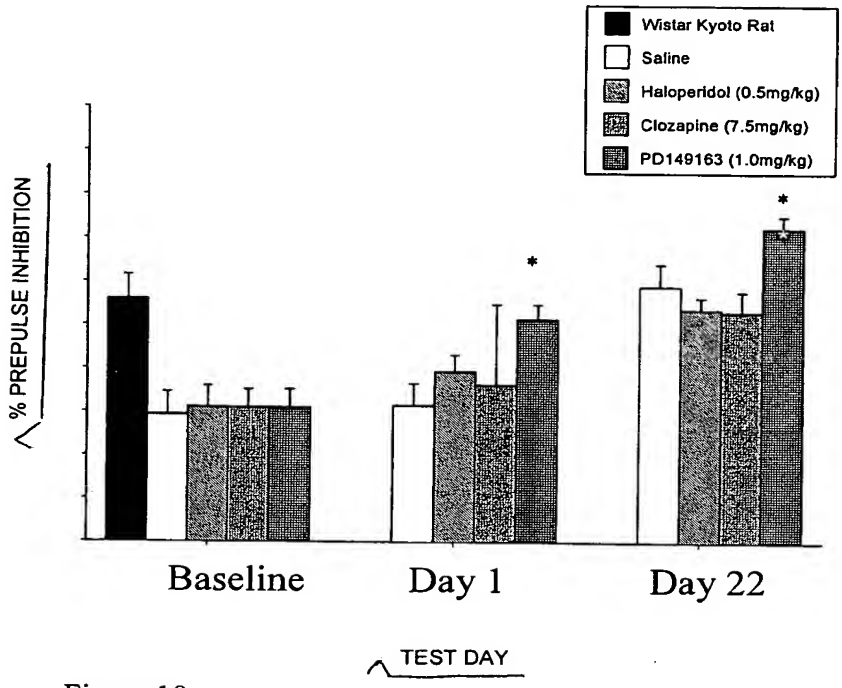

Figure 10

PD149163 but not haloperidol or clozapine increases PPI in BN rats